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Volume XVII NOVEMBER 1956 Number 11

# DISEASES OF THE NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

CONTENTS:

PRACTICAL APPROACH TO ANXIETY AND ITS CONCOMITANT SYMPTOMS IN BRIEF PSYCHOTHERAPY 343
Irving M. Rosen

APNEA IN SUCCINYLCHOLINE-MODIFIED ELECTROTHERAPY ..... 347
David J. Impastato

MANAGEMENT OF THE SIDE EFFECTS OF RESERPINE AND COMBINED RESERPINE-CHLORPROMAZINE TREATMENT ..... 352
Nathan S. Kline, Joseph Barsa and Ernest Gosline

RELATIONSHIP OF ABILITY TO VERBALIZE TO TOLERATION OF LONG TERM HOSPITALIZATION ..... 359
Isabelle V. Kendig, Sol Charen and Louis T. Lepine

RITALIN AND SERPASIL IN THE TREATMENT OF PARKINSON'S DISEASE ..... 363
Leonard I. Lapinsohn

BOOK REVIEWS ..... 366

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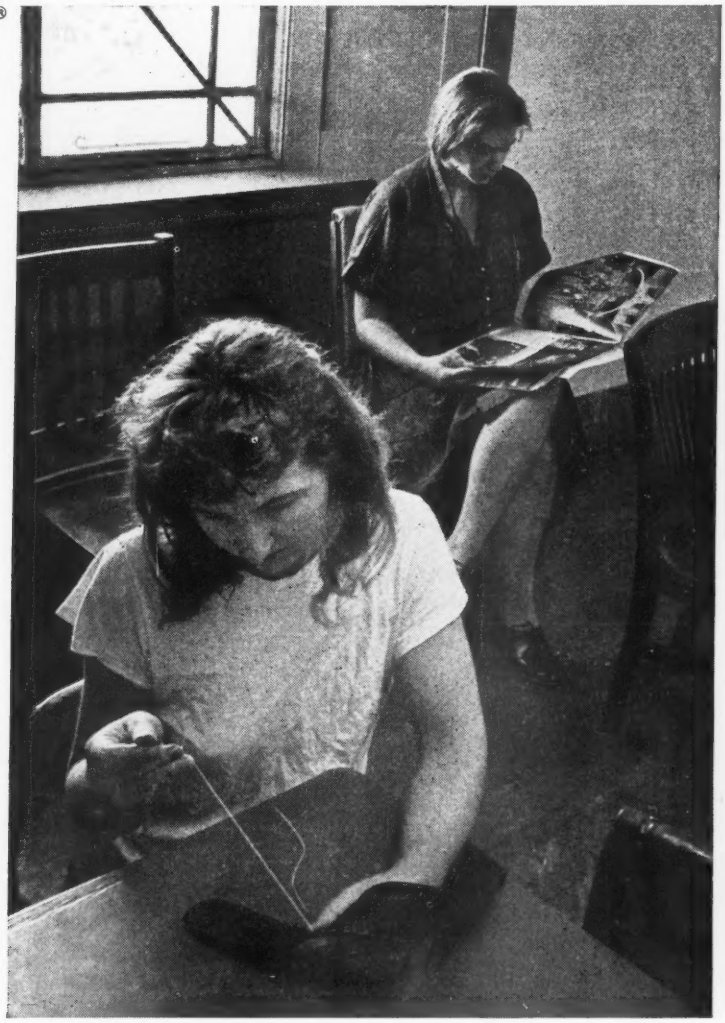

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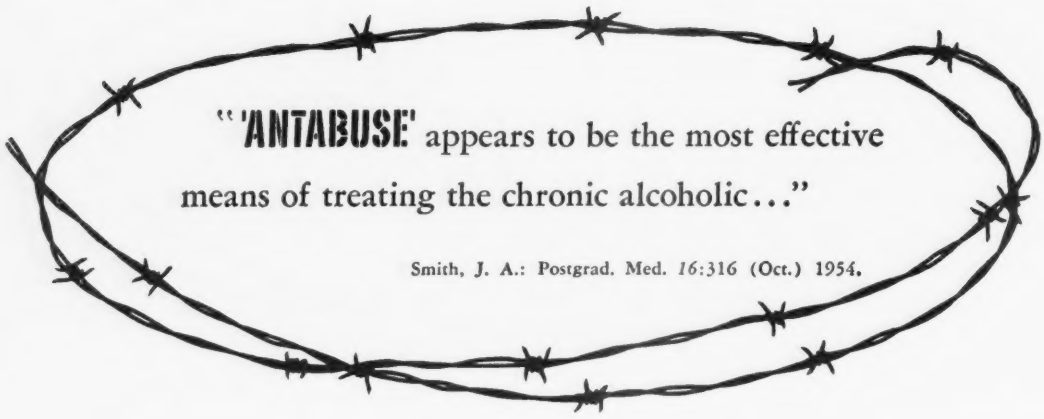


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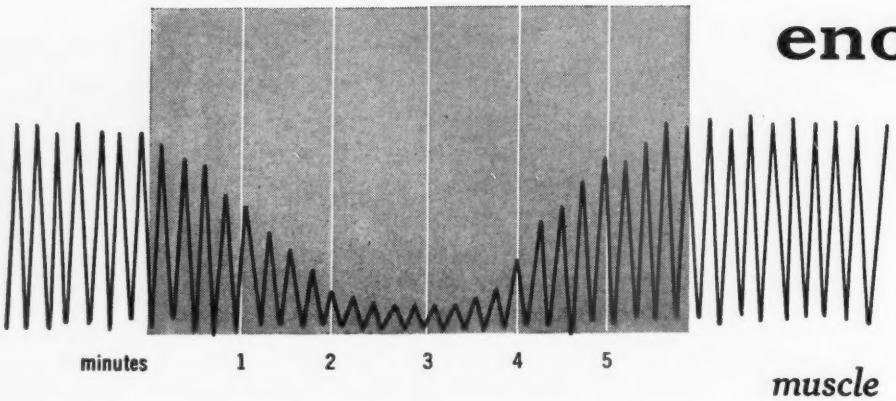


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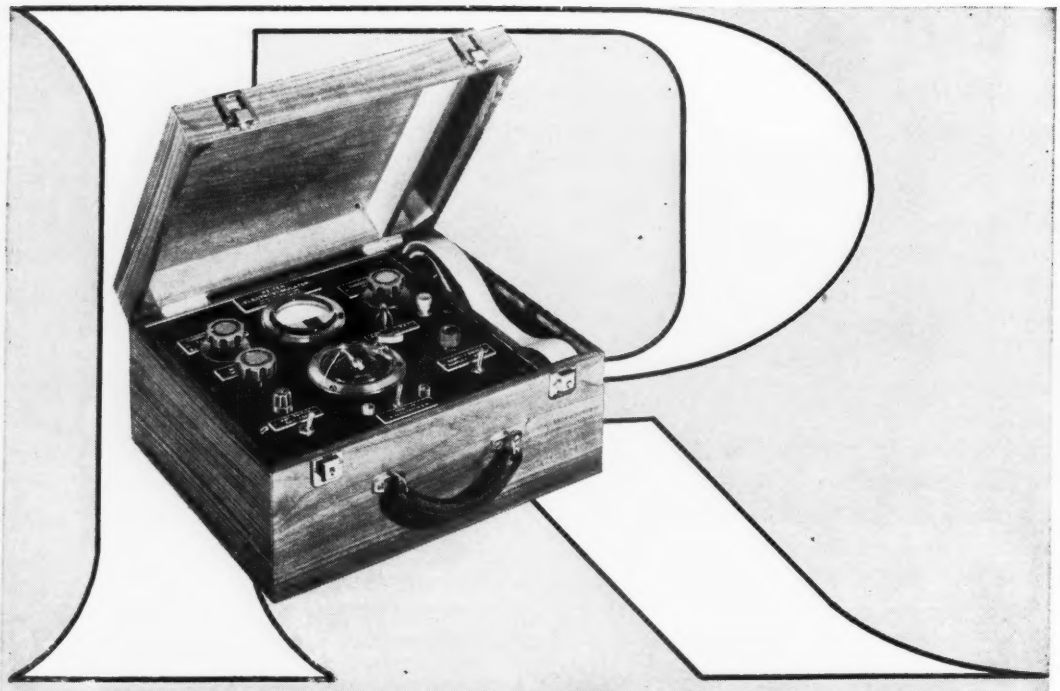

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DISEASES *of the* Nervous System

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

# Practical Approach to Anxiety and its Concomitant Symptoms in Brief Psychotherapy

IRVING M. ROSEN, M.D.
*Philadelphia, Pennsylvania*

This subject, because of its importance, is one of the most commonly encountered in medical literature. This relieves me of the chore of describing anxiety, of distinguishing it from fear, and of discussing theories of its origin. For the purpose of this paper, anxiety is assumed to be a signal to the ego that something in the external world or within the person is wrong and must be restored to homeostatic normalcy. A large percentage of the people whom I have seen in my practice have, on the surface at least, been afraid of anxiety itself rather than of sexual or aggressive excitations. This creates the situation, discussed by Jerome Frank[1] in 1946, in which symptom is etiology, where anxiety itself is one of the issues creating the symptom.. A precipitating event occurs in a predisposed individual and anxiety develops along with a remarkable variety of physical and mental concomitant symptoms. Then this anxiety creates its own trouble. Fenichel[2] states that the anxiety originally intended to prevent a traumatic state actually induces one, and Freud's statement is often quoted[3] that the original source of a fire does not always cause the greatest damage. To make matters worse, each associated symptom sets off a traumatic state of its own. Meanwhile, the beleaguered ego is diverted from solving its original problem.

We must, therefore, help the patient with the distracting element of anxiety. This is a specialized problem and it is not strange that the patient often cannot solve it without expert help. But the patient may surprise us by proving he can handle problems that are not so specialized once he has some idea of what is happening to him. In fact, it may constitute an insult to the ego of the patient for the doctor to assume that the patient is poor in his interpersonal or sexual relations when his ego may, in reality, be having trouble with the problem of anxiety, a medical problem.

What happens to the patient when he develops anxiety? First of all, the very onset may be traumatic. Feelings are bizarre yet fascinating and they may occur in odd places; for example, in a barber's chair or on a streetcar where the victim is doing nothing. Patients often say, "What I am worrying about can't have anything to do with my symptoms because they come on when I am not worrying." I have recently seen two patients with severe continuing anxiety states who were ruminating about the onset of their illness. In one case, the patient felt he had lost his memory while driving a car; the other patient had suddenly felt helpless and had lost her sense of orientation in space. Neither could overcome his dread of recurrence of the symptoms of onset. Also the problem of ego control quickly asserts itself; the patient cannot prognose his ability to prevent himself from carrying out noxious acts.

Intimately associated with anxiety is a wide variety of possible physical and cerebral discomforts. There is not nearly enough time to discuss all the somatic symptoms which seem to emanate from muscles, G.I. tract, and cardiovascular system. It is well known that these symptoms lead to secondary worries including fears of disease. The patient may act on these fears and try to

From Philadelphia State Hospital.
Read in the Section on Private Practice at the 112th annual meeting of the American Psychiatric Association, Chicago, May 1956.

cure himself, for example, he may severely limit his activity in case of fancied heart trouble. Preoccupation with these symptoms may then on a third level cause constriction of social life and in fact withdrawal which obviously will have further bad consequences. Certain somatic symptoms centering about body orifices are particularly mysterious to patients. For example, one pious woman, to her great dismay, invariably developed sensations around the anus when she entered church.

The brain as a source of concomitant symptoms interests me very much. It is an organ with many functions, any, a few or all of which may be disturbingly stimulated by the anxiety signal. Some people fear particularly these cerebral symptoms, others fear more the somatic ones. I have seen psychiatrists and psychiatric nurses thrown into sticky anxiety states by their fear of symptoms which appeared to them similar to disturbances of schizophrenics they had seen or read about. On the other hand the insurance agent whose company pays off for physical ills will fear symptoms that suggest fatal maladies. I do not dismiss the subject of choice of symptoms so glibly. There are, of course, a great many experiences during the individual's life history which predispose him to one symptom or another and especially is it important to consider a history of illness or death to early important figures.

The brain may be stimulated by anxiety selectively, just as it may be stimulated by certain drugs in this fashion. Thus mescaline appears to evoke hallucinations; cortisone affects the mood; lysergic acid plays tricks with the thinking processes, space and time orientation, and the body image. It is instructive to consider some of the things the brain does and then to determine if these activities were increased, decreased, or distorted by anxiety. Emotions may be excessively controlled or labile or distorted to the point where anxiety emerges as doubt or aggressiveness. Auditory, visual or olfactory perceptions may somehow be affected to the point where the patient has hallucinations. I have seen two patients otherwise nearly normal who had the isolated symptom of hallucinations which seemed to go with a state of heightened anxiety rather than with a schizophrenic syndrome. One of these persons responded quickly with a feeling of relief and loss of hallucinations to a straightforward explanation of what anxiety could do to her and was able within the first hour to bring forth the precipitating situation. Perhaps such cases should be called pseudoschizophrenic neuroses. Patients complaining of space and time disorientation and of sensations of depersonalization may likewise be too quickly diagnosed schizophrenia. Symptoms alone should not determine diagnosis; life history, the appropriateness of symptom to situation, and the amount of insight and judgment are more important, not only for diagnosis but for prognosis.

The brain sees connections and associations and this function may be increased or decreased leading either to feelings of excessive coincidence in the environment or of unfamiliarity in common objects. I agree here that patients who complain of such symptoms are usually psychotic. The image of one's body, the level of aspiration and self evaluation appear at times to be distorted leading to secondary anxiety and inappropriate plans. Even one's image of the other person is affected leading to contempt or awe, excessive concern or disregard toward others. The brain mediates memory and thinking which when affected give rise to bizarre feelings and interfere with problem-solving. Thinking by the way involves such activities as abstraction, cognition, experimentation, concept formation, evaluation, use of imagination and foresight. Many patients harbor morbid thoughts; one was haunted by the thought that she would kill her child. This need not be considered necessarily an expression of a wish or of hostility to the child but rather a self torturing idea present to make anxiety more urgent. The safest interpretation might be to tell the mother that she is merely being stimulated by anxiety, that the best way to cause herself more anxiety is to think about the harm that may come to someone she loves. Of course in the back of this morbid thought may be some unconscious hostility toward the child but on the other hand the hostility may be displaced from such objects as husband or mother.

It would considerably impoverish our understanding of anxious patients if we forget the religious and moral functions of the brain. I have been interested recently in listening to patients talking about loss of feelings of purpose, the sudden blotting out of what appears to be a teleological function. Some say a light has gone out inside or life has lost its meaning. One psychiatrist complained that all experience had for him lost its worth and value. In his fright he tried constantly but unsuccessfully to recapture with his intellect the missing feelings. He had been diagnosed obsessive-compulsive and had for periods of months withdrawn into his home. The explanation that a blotting out of value feelings was an associated symptom of anxiety was the most helpful interpretation given during his treatment, so helpful indeed that his diagnosis was most likely not obsessive but anxiety reaction. Sometimes the moral life of the patient is selectively disturbed, in which case he talks relevantly and appears normal but is not trustworthy to himself or others. When this occurs, his spouse may be compelled to act as his conscience. This is usually done in an atmosphere quickly muddled by fears of domination and role reversal and accusations of mental illness which the patient denies, with an accumulation of many secondary social problems. The ego of the patient can be evoked as helper by studying with him what happens when he tries over a set time to live strictly up to the dictates of his conscience. In other words we take the position objectively at the outset at least that his conscience may be sick, not blameworthy. It is possible that a part of the success of Alcoholics Anonymous can be attributed to its symptom-directed techniques aimed at buttressing a weakened conscience. Contrariwise the functioning of the conscience may be accelerated by the presence of anxiety with resulting excessive feelings of guilt and responsibility.

It is not difficult to see how a patient afflicted with the above-mentioned symptoms might become involved in one vicious circle after another. The more intelligent he is, the more he will fear insanity or helplessness or a brain tumor. Some patients feel that their brains must simply wear out from its constant harassment. Others spend much time testing themselves. Many keep asking, Why, why did this happen to me; why does everybody else look so healthy? And every time they ask the question, Why, they get another dose of anxiety. Reading the Book of Job might help if that book were easier to understand. New vicious circles begin when the wife has to go to work or when the patient starts to drink or when he develops an extreme sensitivity to all bodily sensations, even those that are associated with posture and hunger. He has withdrawn from social contacts and has now many little phobias, developed to avoid situations which remind him of the onset of his illness. This finding agrees with the opinion stated by Lief in a recent article[4] that sensory associations linked to the place of the onset of anxiety may explain some phobias. He is getting involved with a variety of doctors and treatments and possibly with hospitals. He has new financial woes and has forgotten some of his special skills. The original problems which precipitated the whole mess remain unsolved. Sometimes special vicious circles necessitate hospitalization or organic therapy. A mother, highly disturbed by noting her neglect of her family, may need to be removed from the home. The shame of being in a mental hospital or fear of the other patients, by constantly reinforcing the neurosis may occasionally stymie psychotherapy and force some other kind of intervention. But for some people, the competitors and the envious ones, organic treatment sets off its own vicious circles by suggesting to the patient that he has a more serious illness than he thought he had, or by making him feel stupid or anergic.[5]

To give the patient the knowledge of what anxiety may do to its victim by serving as its own cause is to take a long step in the direction of helping him quickly. We should guard against the danger of diving too soon below the obvious surface complaints in an effort to seek out etiology. The more we know about the brain's manifold duties, the more interest we will have in the patients' present complaints. This means that the psychiatrist would do well to brush up on his knowledge of descriptive or structural psychology including the psychology of religion,

body image and self, thought and perception in addition to his knowledge of the psychology of ego defenses. And he should also anticipate the consequences and symptomatic pictures possible with diminution, heightening, and distortion of these mental activities. I am not unmindful of the calculated risks involved in paying attention to symptoms. In an important paper Jerome Frank points out six dangers in symptomatic treatment before coming to the conclusion that such treatment is useful not only palliatively but as an aid to a more fundamental approach. The pitfalls of symptomatic therapy noted by Frank are as follows: The therapist may make the patient too comfortable with his symptoms or prescribed drugs to motivate change; he may neglect underlying issues; he may fall into a trap and be led away from the patients' main problems, thereby losing the patients' respect; he may confuse the patients by giving pills for psychic ills.

Not all of the symptoms are unpleasant or evocative of trauma. In fact it would be traumatic to attack prematurely those symptoms which are pleasant, useful, or give the patient a hobby with which to occupy himself, or those which are appropriate and in a sense normal for certain real trap situations. And there must be a reason not only for the precipitation of the anxiety syndrome but also for the patient's present susceptibility to it.

How can the therapjst help the ego to master its own anxiety signal? Quickly, we can attempt to eliminate the possibility of other illnesses, we can explain what we know about anxiety itself, and we can give certain kinds of advice, such as whether to rest or increase activity, to avoid or mingle with people, to get to a hospital or stay home, to take drugs for relief or not. While we might make a diagnosis, it is more important that we evaluate for the patient his ego control, that we try to estimate whether he will really faint or become insane. Should he continue to drive his car or not? Do we think the mother will kill her baby or not? The chances are that if the patient has not lost control up to now, he won't.

It is important and often forgotten that even in brief psychotherapy there is a kind of working through since the patient will have to discuss each one of the anxiety concomitants that worry him. During all this ego buttressing, aspects of the patient's original precipitating and predisposing problems will emerge and become available for solution.

Mental illness appears to spread within the individual in three ways: by horizontal increase in the number of anxiety concomitants, such as palpitation, anorexia, fatigue, or interference with thought; by accretion of new problems brought about by the original symptoms or the patient's attempts at self-treatment; and by retreat of the ego, as Karl Menninger[6] recently conceptualized it, behind increasingly maladaptive defenses.

*Summary*

Fear of anxiety itself is the commonest presenting cause of anxiety, much more common in my suburban practice than fear of sexual or aggressive excitations. Each sensation or discomfort associated with anxiety causes its own traumatic state. Indeed the very mode of onset of the condition brings its own peculiar trauma. When anxiety gives rise to bodily symptoms, the doctor is not likely to misinterpret them; but when the brain is the organ responding with symptoms, the therapist may miss their import and mistake an anxiety reaction for a more serious condition.

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# Apnea in Succinylcholine-Modified Electroshock Therapy

## (A New Technic of Succinylcholine Administration Without Barbiturates)

DAVID J. IMPASTATO, M.D.
*New York, New York*

*I. Introduction*

Electroshock therapy (EST) has established itself as a specific treatment for moderate to severe psychotic depressions. It is also a potent modality for any psychosis of acute onset. In attempts to make this treatment safer and more acceptable to patient and physician, a number of modifications have been made,[27,32,33,34] the most recent of which is succinylcholine dichloride (SCC), a muscle relaxant of brief duration which so effectively reduces the muscular force of the electroconvulsion that, with its use, fractures have been almost entirely eliminated. Additionally there is a marked diminution or complete elimination of convulsive movements and with administration of oxygen during treatment the work load on the heart is so reduced as to make it relatively safe to treat even poor risk cardiac patients. The administration of SCC carries with it practically no risk except for apnea which in most cases is inconsequential, but in some instances may be very prolonged and might even lead to death.[31] In view of this, knowledge of the causes of this type of apnea, its prevention, and its management are essential for the proper administration of this valuable medicament.

*II. Causes of Apnea in Succinylcholine-modified Electroshock Therapy.*

*A. Factors Which Render the Patient Especially Sensitive to Succinylcholine.*

SCC in moderate doses usually causes apnea of short duration from temporary paralysis of the intercostal muscles and diaphragm. Large doses may cause prolonged apnea of varying duration. The response of the patient to the drug is due to a number of factors such as high blood potassium,[1] fever,[2] and the amount of circulating adrenalin.[3] These enhance the effect of SCC. Response is also greatly dependent on the pseudo cholinesterase in the plasma;[4] a low titer prolongs and intensifies the effect of SCC.[5] Low titers may be found in severe anemia, liver diseases, cachexia, poisoning with anticholine esterase substances, such as nerve gasses and some weak killers, Parathion and T.E.E.P., etc. It follows that such conditions be suspected and detected whenever possible and smaller doses of SCC used on these patients. Low pseudocholine esterase has been found in about one per cent of all patients;[5] and in a much larger percentage of patients showing prolonged apnea.[6] Although the presence of these factors determine the response of the patient to SCC, in general the same dose usually produces the same degree of muscular weakness in the same patient. We have noted, however, that in some patients a moderately high dose is required to produce the same effect as the treatment progresses. This observation has also been made by others.[16]

Prostigmin (neostigmin) and physostigmin (eserine), which are antidoes for d-tubocurarine and other curare derivatives, are not antidoes for SCC; in fact, they prolong its effect by inhibiting the plasma pseudo choline-esterase, and should not be used to combat apnea caused by SCC.

*B. Dosage and Rate of Administration of Succinylcholine.*

There is no schematic way to properly estimate the effective and safe dose of SCC, and we agree with Steven et al.[17] who emphasize that "there is no fixed dose of SCC that can be used on all patients." Estimates based on the weight, the muscularity, etc., of the patient, as well as standard doses given to each patient regardless of age or weight, are unphysiologic because the response of the patient to the drug depends on many factors other than weight, muscularity or age. Possibly the best way of estimating the therapeutic dose of SCC is to give the patient a small 5 mg. test dose (see below) and then estimate the therapeu-

tic dose on the response to the test dose. Many serious apneas will undoubtedly be avoided if this precaution is taken.

Some workers administer SCC in 5 to 10 seconds;[15,25] while others in from 30 to 40 seconds or longer.[26] When the SCC is given quickly, a much smaller dose is needed to produce the same degree of muscular relaxation; when the SCC is given slowly, a larger dose is required to produce the same degree of relaxation and, in addition, the larger dose carries with it a greater risk of producing a prolonged apnea.

A larger dose of SCC causes a longer apnea. Day[6] found that with 50 mg. of SCC, apnea lasted 7½ minutes; with 40 mg., six minutes, and with 20 mg., three minutes. A similar observation on the direct relationship between the dosage of SCC and the duration of apnea was shown by a patient in whom 30 mg. of SCC caused apnea lasting five minutes, 20 mg. 2½ minutes, and 10 mg. two minutes.[11]

Glover and Rosium,[15] using SCC doses of from 5 to 20 mg. without barbiturates, had only one instance of apnea lasting five minutes, which occurred in an alcoholic with an enlarged liver. Murray,[14] also using small doses of SCC without barbiturates, had no apnea lasting longer than one minute, 45 seconds in 5/3 patients. In contrast to this, Lewis et al.,[24] who "aimed at complete paralysis," using 20 to 80 mg. of SCC patients had periods of apnea lasting from 1½ to 11½ minutes and in over 70 per cent the apneas lasted from 3½ minutes to 6½ minutes. These patients were also given an ultra-short acting barbiturate in relatively large doses. This illustrates vividly the tendency of large doses of SCC to produce prolonged apnea.

*C. Concomitant Use of Ultra-short Acting Barbiturates with Succinylcholine.*

Most workers use ultra-short acting barbiturates with SCC. As is well known, barbiturates are strong respiratory depressants and capable of causing prolonged apnea,[12,13] especially when used in large doses.[24] There is danger in compounding a SCC apnea when barbiturates are used. For this and other reasons, Murray,[14] Glover and Rosium,[15] and others advise against the use of barbiturates with SCC. It must be said, however, that barbiturates have been extensively used with SCC and small doses have, in the main, caused little difficulty ,especially when these have been combined with small doses of SCC. The use of short acting barbiturates with SCC should not be discouraged, as they are helpful in the management of extremely anxious patients, and in patients who develop anxiety following SCC modified EST. Until recently I have used small doses of barbiturates in most cases without encountering any difficulty.

*D. Sensitive Respiratory Center.*

It has been shown in animals that, when large doses of SCC are given, the respiratory center is paralyzed. In cats, which developed protracted respiratory failure following large doses of SCC, muscular tone returned before respiration; and recording of the action potentials of the phrenic nerve showed absence of stimuli from the respiratory center.[7,8] Although suppression of the respiratory center is in most cases due to overdose of SCC or indirectly to the presence of sensitivity factors, the possibility still exists that, in some patients, the center itself may be sensitive to SCC in the absence of sensitivity or overdose. This may explain the prolonged apnea produced by 4 mg. of SCC as reported by Robie.[23]

*E. Improper Oxygenation.*

Apnea may be prolonged by lack of oxygen and the resulting anoxia may produce cardiac arrhythmias and cerebral damage.[24] The proper administration of oxygen with controlled assisted respiration, either with a resuscitator or manually, is essential to the successful management of patients with apnea, and especially those with prolonged apnea.

Methods of oxygen administration during SCC modified EST vary.[26] Some investigators oxygenate the patient before, during, and after the EST; others oxygenate the patient as soon as the SCC has been given, continue until the convulsion is initiated, and resume it at the end of a convulsion. Most observers administer the oxygen at the end of the convulsion. We have found the latter method the most convenient. When oxygen

is given prior to the convulsion and during the convulsion, the fit is unduly prolonged. This may be disadvantageous, especially when little modification of the fit has been obtained. With patients in good physical condition, there is no necessity of administering oxygen until the convulsion is over; on the other hand, in poor risk cardiac patients as well as in pregnant women, administration of oxygen prior to as well as at the end of the fit is of benefit.

*III. Prolonged Apnea*

Any apnea lasting more than five minutes may be considered prolonged. Prolonged respiratory failure following SCC lasting from ten minutes to three days has been observed by a number of investigators.[4,5,10,16,18,19,20,21,22] Invariably these prolonged apneas have occurred in patients who had received large doses of SCC, from 20 to 80 mg. However, a few patients developed apnea following a small dosage—4 mg.[23] Most prolonged apneas are not necessarily dangerous, provided the patient is properly oxygenated throughout the apneic period. Adequate oxygenation in these patients is easy due to the completeness of the paralysis which precludes laryngeal spasm. This, however, is only for the first few minutes of the apnea or until muscular power has returned, at which time laryngeal spasm may occur and proper oxygenation may not be maintained in some patients without an endotrachial airway. However, it must be noted that intubation has been rarely needed following SCC modified EST. Prolonged apnea is best avoided by using small doses of SCC.

Recently, Dr. Richard D. Kepner of Honolulu reported to me that a patient suffered from a 20 minute apnea following 20 mg. of SCC administered by another physician on the occasion of the first shock treatment. Had this patient been pretested with 5 mg. of SCC, this apnea may have been avoided. Some investigators use routine doses of 20 mg. or more of SCC on all patients. If this had been Robie's[23] practice, his patient who developed a severe apnea following 4 mg. of SCC would probably have died. It is strongly recommended that all patients be tested with 5 mg. of SCC prior to the administration of the first EST.

*IV. Treatment of Prolonged Apnea*

In the presence of prolonged apnea, the following procedure is recommended:

1. Administer atropine, 1.2-2.4 mg. (1/50-1/25 grain) intravenously.
2. Oxygenate the patient properly, preferably with a positive-negative resuscitator.
3. Ascertain whether respiratory failure is central or peripheral. Muscular movement or the presence of tendon reflexes indicate that the peripheral mechanism is in order and that the apnea is central; while absence of these signs implicates the peripheral mechanism—diaphragm and intercostals or airway.
4. In the presence of paralysis of the respiratory center, administer lobeline 10 mg., or coramine 5 cc, or metrazol 5 cc, intravenously. Electric stimulation with the unidirectional machine to induce respiration may be tried by placing the two electrodes on the temples near the front of the ears and applying very brief bursts of current up to 10 ma. for a second or two. This may be repeated several times until the patient begins to breathe.
5. If the paralysis is peripheral, give whole blood or a concentrate of plasma pseudocholine esterase intravenously.
6. Estimate serum potassium and sodium. If a high potassium level is found, administer sodium lactate 1000 cc of 1/6 molar solution, intravenously. If potassium level is low, administer 0.3% potassium chloride solution at a rate of 100-120 drops per minute.[29]

If EST is to be resumed in a patient who has suffered a prolonged apnea, proceed by omitting the barbiturate altogether, increase the atropine and reduce the dose of the SCC to ½ or less of the dose that produced the apnea. If the patient has not been tested previously with the 5 mg. test dose of SCC, this should be done and the therapeutic dose based on the test response.

*V. Our Method of Administration of Succinylcholine in Electroshock Therapy.*

We have given SCC to approximately 200 patients without encountering prolonged apnea, fractures or other complications. Our methods of treatment are as follows:[25,26] At the time of the first treatment, a test dose

of 5 mg. of SCC is given without any other drug to test the patient's muscular and respiratory responses.[26] When properly managed, the patient does not develop anxiety from this test. Usually, the respiratory and muscular responses to a dose of SCC are of equal degree. However, in a few patients a discrepancy occurs and there may be a severe respiratory paralysis with little muscular paralysis, or vice versa. If, there is a strong respiratory depression with little muscular response, the therapist must choose between a possible prolonged apnea if he gives a dose sufficient to cause profound paralysis, or risk a possible orthopedic trauma with a dose causing insufficient muscle weakness. I always choose the latter course because the danger of fracture under these circumstances is small, while the danger of prolonged apnea with possibility of a disaster[31] is greater.

According to the test response, the patient receives the smallest possible therapeutic dose of SCC to achieve the degree of muscular paralysis required which dose is maintained, lowered, or raised as necessary on subsequent treatments. Our dosage of SCC has varied from 5 to 20 mg.—with most patients receiving from 10 to 15 mg. At the first treatment, the estimated therapeutic dose, in the same syringe containing 0.8 mg. (/75 of a grain) of atropine sulphate, 50 to 75 mg. of thiamylal (Surital) or 100 to 150 mg. of thiopental (Pentothal), is injected quickly within five to ten seconds, two to three minutes following the test dose. Within ten to fifteen seconds following the therapeutic SCC injection, the EST is given. At the end of the clonic phase oxygen under pressure is given until the patient is breathing spontaneously. We have aimed at only a 50 to 75 per cent reduction of muscular power rather than a complete paralysis as we feel that this is sufficient to avoid practically all fractures. Using this technique, we have had only a few apneas lasting more than two minutes. In many patients, there was no need of oxygen as they breathed spontaneously following the convulsion. This experience matches that of other workers who also use small doses of SCC.[14,15,23] In all our patients we rarely had to use the oral airway and have never had recourse to intubation. Although no fractures occurred in our patients, Murray[14] in 573 patients, reported a fracture following 10 mg. of SCC given to a patient with chronic rheumatoid spondylitis, and Glover and Rosium,[15] in several hundred patients, had an infracture of the neck of the humerus. In patients with existent fractures, osteomalacia or spondylitis, we have not hesitated to give SCC in doses sufficient to cause a more or less complete paralysis and have assumed the risk of prolonged apnea. None has occurred in the few patients we have so treated.

In debilitated, chronically ill patients, in alcoholics, and those with liver disease, treatment should begin with smaller doses of SCC than estimated and also possibly with a unidirectional machine so as to produce a soft convulsion. The dose of SCC can be subsequently adjusted to the response of the first treatment.

Recently I have begun to use SCC without barbiturates as advised by Murray[14] and others. I formerly had used SCC with barbiturates[25,26] on the assumption that all patients treated with SCC without a barbiturate developed anxiety.[10,17,24,30] Apparently this is not so, and many patients can be successfully treated with SCC alone.[14,15] The omission of the barbiturate minimizes the possibility of prolonged apnea and removes the possibility of side effects of the barbiturate itself, such as a pulmonary edema, vomiting, aspiration, pneumonia, etc. A disadvantage of SCC alone is that, if not properly administered, the patient will become anxious and possibly refuse further treatment. In addition, there is also moderate increase in post treatment restlessness.

More recently I have developed a new technic of administering SCC without using barbiturates. The disadvantages of administering SCC without barbiturates are: (1) the patient may develop anxiety if the EST is not given within ten seconds or so from the end of the SCC injection, and (2) the interim is insufficient to allow full paralytic effect of the SCC to develop. I have overcome both of these disadvantages by injecting the SCC without barbiturates in the usual manner, waiting ten seconds, then giving a petit mal stimulation, which does not produce a convulsion, but does effectively ob-

literate the memory. Twenty to thirty seconds after the petit mal the grand mal is given. This technic produces complete amnesia without anxiety, full paralytic response with minimal doses (10 mg. or less) of SCC and practically no apnea.

*VI. Summary.*

1. Apnea is a serious problem in electroshock therapy as it might result in a fatality.

2. It is enhanced by barbiturates.

3. The larger the dose of succinylcholine chloride, the greater the danger of apnea.

4. Prolonged apnea is usually caused by high doses of succinylcholine.

5. Factors which render patients especially sensitive to succinylcholine have been discussed.

6. There are two schools of thought regarding succinylcholine administration. One school believes in anesthetising the patient and using large doses of succinylcholine to produce complete muscular paralysis; a procedure often accompanied by prolonged apnea. The other school believes in small doses of barbiturates or no barbiturates at all, and small doses of SCC, producing only 50 to 75 per cent reduction of muscular power. With this method, serious apnea is very rarely produced. We recommend the latter method.

7. Our methods of treatment and a new technic in administering succinylcholine without barbiturates are described.

8. A test dose of 5 mg. of succinylcholine at the time of the first electroshock treatment is strongly advised for all patients.

9. An outline of steps that may be taken in the treatment of prolonged apnea is given.

10. Recommendations are made as to how to proceed following a successful outcome of prolonged apnea.

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*Author's Note:* Succinylcholine dichloride (SCC) used in this study is manufactured by Burroughs Wellcome & Co., Inc., under their trade name Anectine and was generously supplied by Dr. William P. Colvin.

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# Management of the Side Effects of Reserpine and Combined Reserpine-Chlorpromazine Treatment

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The widespread use of reserpine and chlorpromazine, either individually or in combination has brought to attention the need for handling the side effects which these drugs sometimes induce. The greatest problem in this respect is not in the state hospitals where the side reactions constitute, as a rule, nothing more than annoyances. It is in private practice that some of the concurrent reactions make it extremely difficult for the patient to function adequately in his usual setting. Because of the frequent use of these drugs by general practitioners for minor psychiatric conditions, it is important to present the material on the management of side effects gained in the treatment of many thousands of such patients. The extent to which the drugs are currently being used to replace barbiturates is indicated by the fact that in one of the major pharmaceutical houses the sale of barbiturates has dropped off approximately one-third in the past two years and the anticipation is that the drop will continue.

The ideal method for managing side effects of a drug is to replace it with another drug which does not have side effects. We are currently working with a number of preparations which it is hoped may eventually provide substitutes for the present medications and at the same time be equally effective. Up to the present, however, evidence is not conclusive that any of these preparations will do this. We are probably at much the same stage that we were in the treatment of infections at the time when prontosil, sulfanilamide and sulfathiozole were the methods of choice. These have all been replaced by more effective and less toxic preparations and although it is probable the same will occur with the current compounds, there may be a lapse of several years and it is therefore felt justifiable to present our experiences until more efficient pharmaceuticals are available.

We are currently completing a number of studies to help clarify the indications for reserpine, chlorpromazine, and combined reserpine-chlorpromazine. We felt that it would be unwise to present a summary of the

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toxic effects of the two drugs within the confines of a single paper, since the implication might be drawn that one drug was to be preferred over the other because of differences in side effects. This is certainly a factor in determining the choice of medication but this cannot be considered by itself and until we are also able to give the positive indications with some degree of certainty, it seemed wiser to confine ourselves to the drug with which we were more familiar and had had the longer experience. We have included a brief mention of the combination of reserpine and chlorpromazine but this is because we believe that the mode of action is primarily that of reserpine (with chlorpromazine acting as a potentiater). The difference between reserpine alone and combined reserpine-chlorpromazine are also referrd to in this paper.

In presenting the side effects, we have divided these into organic and psychological and within each group have in a general way listed the side effects in order of their frequency. We have not attempted to give statistical or percentage approximations of the incidence of each side effect but to lay stress upon the management of the symptoms when they do occur. Undoubtedly, there exist other side effects which we have not described but it is our feeling that their relative frequency is not great, and the following descriptions should prove adequate in the great majority of patients.

### *Physical Side Effects*

1. *Generalized Tremulousness.* This is distinct from the typical parkinsonism, which is described later. Whether it is of extra pyramidal origin remains an open question but certainly the large majority of patients do suffer from this symptom. In the management of this condition, we have found benztropine methanesulfonate (Cogentin, manufactured by Sharp & Dohme) to be the drug par excellence in the handling of this condition. One mg. twice a day is our usual dose. The drug seems so effective in this respect that we ran a separate experiment in which it was demonstrated that benztropine methanesulfonate, in a series of 15 patients, did have a definite sedative effect when used by itself but did not bring about the psychiatric improvement which occurs with both reserpine and chlorpromazine. We also have used glutethimide (Doriden, manufactured by Ciba Pharmaceutical Products) in a number of cases. It was demonstrated that this drug, too, was effective in the management of this side effect but that the benztropine methanesulfonate was superior.

2. *Hypotension.* In the original study,[1] it was demonstrated that even with small doses of one mg. that reserpine tended to lower blood pressure in hypertensives, normotensives and patients already hypotensive. In most cases, even though systolic and diastolic pressures were extremely low, the pulse pressure maintained itself remarkably well. In patients in whom the blood pressure dropped to a point where the physician (in contrast to the patient) began feeling concerned, or in the rare cases where the hypotension did produce disabling effects, we would recommend the use of one of the analeptic drugs. (Drinalfa, Squibb trade name for desoxyn; Dexedrine, Smith, Kline & French trade name for dextro-amphetamine sulfate; Ritalin, trade name of Ciba Pharmaceutical for methyl-phenidylacetate; Meratran, Merrell trade name for pipadrol hydrochloride). In cases where these did not prove effective or if there were contraindications to their use, we have also found the anticholinergic drugs at times equally effective. A combination of 1/100 gr. atropine and 3/8 gr. of ephedrine used TID is also useful.

3. *Drowsiness* is also extremely common and here the same stimulant preparations have proved effective. In occasional cases glutethimide has seemed to relieve some of the drowsiness but in this respect it is inferior to the analeptics.

4. *Flushing* is common on the initial use of the drug, especially if the intramuscular preparation is used. This does not warrant any special handling since, after the first few doses, the flushing no longer occurs. This is a normal pharmacological reaction.

5. *Fatigue,* as distinct from drowsiness, is also common and here again the stimulants are the most adequate way for managing this condition.

6. *Nasal Stuffiness* can be annoying at times and local treatment by means of a

nasal decongestent is recommended. We have used nephazoline. (Privine, Ciba Pharmaceutical Products.) If the condition persists over a long period it is recommended that the decongestents be used with caution (in accord with the statement of the manufacturers) because such preparations may cause a rebound congestion.

7. *Gastro-intestinal Symptoms.* These usually consist of mild cramps, diarrhea, nausea and occasionally vomiting. Under most circumstances these are self-limited but if they persist or are unusually severe it may be necessary to treat them actively. In some cases the glutethimide has proved effective and in other cases the benztropine methanesulfonate (which has an anticholinergic radical) has also served to alleviate or reduce the symptoms. The former drug seems to cause a reduction in the nausea and vomiting without effecting the cramps and diarrhea. If neither of these prove adequate, we have found the best method of treatment to be the anticholinergic drugs which are almost 100 per cent effective in relieving these symptoms. Belladonna, atropine or one of the synthetic preparations all seem equally effective.

8. *Dizziness.* We have as yet found no individual preparation which seems useful in reducing this side effect. One of the drugs known to control motion sickness might be useful. We have not had occasion to evaluate the use of these, so this remains merely a possibility.

9. *Ptyalism.* Some degree of drooling or excessive salivation is commonly found. The anticholinergics including benztropine methanesulfonate have proved most effective. One of us (E.G.) also reports success with glutethimide in some cases.

10. *Muscular Symptoms.* Muscular cramps, twitching, and paresthesias are not infrequent. In some cases these respond to the benztropine methanesulfonate if the symptoms are limited to the cramps and twitching. This drug seems to have little or no effect on the paresthesias. The only preparation which seems to effect the paresthesias at all are the anticholinergics and even there the results are unpredictable and not entirely satisfactory.

11. *Urinary Frequency.* Benztropine methanesulfonate has proved helpful at times in cases where this is a disturbing complaint but the anticholenergics seem the most effective means of controlling this side effect.

12. *Edema.* We[2] have previously estimated that approximately 20 per cent of the patients receiving intensive reserpine treatment develop edema of either the face or extremities or both. Although this may be of minimal degree it may prove disturbing and uncomfortable to the patient. In some cases facial edema seems to subside after antihistaminics have been given and the combination of reserpine with an antihistaminic (such as Sandril with Pyronil by Eli Lilly & Co.) may be useful. The edema of the extremities is not relieved by any specific pharmaceutical that we have as yet tested.

13. *Weight Increase.* Many of the patients on reserpine show a remarkable increase in their food consumption and describe their appetites as "ravenous." This is influenced to some degree by the analeptics but not as satisfactorily as in the control of appetite of patients not on reserpine. This is not always an undesirable side effect and there is a question as to whether any attempt should be made to reduce this particular action of the drug. The concern is usually esthetic although, occasionally, patients keep outgrowing their clothes which may be an economic problem.

14. *Thirst.* We do not feel that the appetite gain is primarily due to increased thirst which occasionally occurs. There is no specific treatment for this symptom.

15. *Parkinsonian Symptoms.* These include all typical stigmata of parkinsonism and may even involve oculogyric crises. The occurrence of this side effect is not an indication for withdrawal or reduction of medication unless the psychiatric condition has improved sufficiently. Both benztropine methanesulfonate and glutethimide help considerably in controlling these symptoms although they may not eliminate them entirely. One of us (J.B.) feels that glutethimide is not as satisfactory. We have not as yet run controlled studies to test the relative efficacy, so that at present it must simply be noted that this is our present experience.

16. *Dermatitis.* Only one of us (N.S.K.) has seen a case which appeared to be an or-

dinary allergic sensitivity. This patient regularly had recurrence of dermatitis when placed on the drug and remission when drug was discontinued. Much more frequent is the occurrence or accentuation of seborrheic dermatitis which occasionally involves the entire body. The antihistaminics seem to be of help in relieving the itching. Promethazine (Phenergan by Wyeth) was particularly effective in this respect. Treatment of the underlying condition with selenium sulfide (Selsun by Abbott) has proved effective in clearing up the seborrhea.

17. *Eye Symptoms.* These may consist of either pupillary dilation or constriction. In occasional cases midriathis has interfered with the functional activity of a person who must either read a great deal or use his eyes for other purposes (in one case an artist was unable to paint satisfactorily). In view of the pharmacology on cats this is a puzzling reaction since it is the opposite of the usual anticipated result. Very rarely one of the analeptics helps to reduce this condition but in a number of cases the simplest solution has been to have the patient fitted with temporary glasses which correct the defect. These can be discarded when treatment is completed.

18. *Impotence.* It is well to know that in males the drugs sometimes act to reduce both the sexual desire and functional capacity of the patient. In cases where it may cause concern it is perhaps wise to indicate to the patient that this is a possibility, since he may become very concerned and reluctant to mention it if he does not know that it may occur as a "natural" side effect. There is no specific pharmaceutical of use and if the condition is not self-limited (as it often is) the patient must patiently or impatiently wait until treatment is completed.

19. *Lactation.* In occasional patients the reserpine will also induce lactation and this is particularly likely to give concern unless the physician is aware that this side effect can occur. There may be gynecomastic and/or pseudolactation in both sexes.

20. *Menstrual Irregularities.* Menstrual irregularities including menorrhagia, metrorrhagia, amenorrhea, etc., occur but are uncommon. No systematic treatment for this side effect has been developed.

21. *Anorexia.* Although infrequent in contrast to an increase in appetite some patients develop anorexia. Occasionally small doses of regular insulin used an hour before meals may help to correct this condition. In severe cases, it is sometimes necessary to discontinue treatment.

22. *Convulsions.* In approximately one per cent of the patients convulsions have occurred during the course of treatment. These are usually limited to a single convulsion during the first six weeks of treatment. No specific medication has been used since the attacks have not been repeated. In a patient with a history of convulsions it might be wise to continue barbiturates during reserpine treatment.

23. *Toxic Syndrome.* It is possible to give an overdosage of reserpine. This usually occurs only at high limits after prolonged administration. Reduction of appetite after earlier increase, and organic type confusion, singly or in combination, may be indication that the drug is having a toxic effect. Treatment in this case is by reduction or withdrawal. Usually reduction will be sufficient to clear up the syndrome. This is usually not confused with the basic condition, since in most cases the patient has shown marked improvement and after a month or more of treatment the toxic syndrome begins to develop.

24. *Midbrain Syndrome.* Very rarely the syndrome appears which involves the midbrain with the development of hyperpyrexia, ocular palsies, decerebrate movement and other symptoms of like origin. Mental defectives appear particularly prone to the development of this condition and should be treated with caution and watched closely for early signs. This probably occurs only when there is pre-existing brain damage, anomaly or deficiency. No treatment beyond withdrawal of the drug is presently known.

25. *Reduced Resistance to Intercurrent Diseases.* Patients being treated with reserpine (and chlorpromazine) appear to have a lowered resistance to disease and require more vigorous treatment than would ordinarily be the case. They tend to develop infections (pneumonic and other types) more easily than patients not under treatment and

larger doses of antibiotics or other medicaments are required. If a cardiovascular condition is present, the patients seem to go into decompensation more easily and in general treatment more digitalis is required than would be the case for similar patients not under treatment. Another evidence of this is the frequency with which patients who are in bed for only a few days tend to develop marked trophic ulcers of the heels. These also are more resistant to treatment than is ordinarily the case. It may well be that some of the fatalities reported with patients on reserpine who were given electric shock may be due to some inability to cope with stressful physiological conditions such as described here. We have no specific means of correcting this condition but merely caution that patients on reserpine who develop intercurrent diseases of any type should be watched more closely. In occasional cases one of us (E.G.) has found glutethimide useful. In some patients where the drug was discontinued because of cardiovascular side effects they were given glutethimide then reserpine was resumed and successfully carried to completion. The mode of action of glutethimide is difficult to explain although the "antagonism" between reserpine and this drug may be due to the fact that glutethimide is related to certain of the imides of glutaric acid, which have been shown (report by the Research Department of Ciba Pharmaceutical Products) to have antiparkinsonian activity.

### *Psychological Side Effects*

The psychological side effects are not listed in order of approximate frequency since it is easier to view them as a number of groups of symptoms rather than isolated occurrences.

1. *Lethargy.* One of the marked beneficial effects of reserpine is that in many cases patients are able to "normalize" environmental situations to which they were hypersensitive. This state can pass from one of relief from external pressure to a type of "belle indifference" and finally to a point where the patient's interest and concern with his environment becomes more reduced than is desired. In most cases these patients respond to stimulants.

2. *Retardation.* The lethargy may be accentuated to the point where patients are actually retarded both physically and mentally. Their actions are slow, they have difficulty focusing on environmental stimuli and they appear somewhat dazed. Here again, the analeptics seem the best method of treatment although they are not so successful as in the treatment of lethargy.

3. *Withdrawal.* At the extreme the patient as a result of reserpine may put such psychic distance between himself and his environment that he is almost out of contact. There is usually no other evidence of abnormal mentation or emotion. The analeptics are only occasionally and slightly of use. With a few patients glutethimide has seemed to relieve this side effect. If withdrawal occurs after the patient has already shown marked psychological improvement, it is recommended that the dosage be reduced.

4. *Restlessness.* Just as some degree of lethargy is evident in practically all patients, the same is true for a certain amount of restlessness. It is difficult to find a precise term to describe how these patients appear to feel but it is evident that many of them are discomforted and do not quite know what to do with themselves. This is true both in respect to psychological as well as physical behavior and not infrequently patients are found pacing about since they have a need to be doing something and they find it difficult to sit still. This is quite different from the rather aimless pacing which some schizophrenic patients demonstrate, since it is done with awareness that it is serving a purpose of relieving some sort of pressure. Both glutethimide and benztropine methanesulfonate have been used successfully although, one of us (E.G.) does not feel that the latter drug is as effective as the former.

5. *Agitation.* The symptoms described above may become accentuated so that the response is more than mere restlessness. Ordinarily the treatment is the same but if severe it is sometimes necessary to add hyoscine. It is worth noting that barbiturates are not very effective.

6. *Turbulent Phase.* We have described the nature of the turbulent phase elsewhere in the literature[3] and this appears to be an

extreme expression of the same side effects described in the two previous items. Patients may become extremely disturbed physically, emotionally and mentally and what was formerly a "quiet" psychosis may become very active. There is irritability and proneness to "act out" which occurs in the neurotic as well as the psychotic individual. There appears to be a lack of awareness of dangers which results in injury-proneness. In the extremes of excitement the patients sometimes require isolation for a short period. Benztropine methanesulfonate is the drug of choice for treatment in these situations. When the patient begins to "come out" of this phase his movement into the integrative period can be expedited by the use of glutethimide (which, however, is not very effective in dealing with the prior and more severe part of the turbulent phase). Both drugs are useful if the "equivalent" reactions occur in neurotics.

7. *Insomnia.* It is not unusual, particularly at the lower doses, for patients to complain of insomnia. Of the preparations which we have described, glutethimide seems to be very effective and as many as three 1 gram tablets can safely be given (if needed) at the appropriate time. This is almost always effective. Benztropine methanesulfonate is effective when the insomnia is due to tension and restlessness associated with the turbulent period.

8. *Dreams and Nightmares.* Dreams are more vividly recalled than is ordinarily the case and not infrequently seem to extend into the day, so that patients sometimes have difficulty shaking themselves free of the experiences of the previous night. Whether there is actually an increase in nightmares or merely that they are recollected more readily is difficult to determine. Not all of the dreams are unpleasant and frequently they are excitingly erotic in nature.

9. *Increased Eroticism.* Not only the dream life but frequently the waking life is also characterized by greater than usual erotic feelings. This is in marked contrast to the patients developing impotence. This obviously can cause social complications at times but there appears to be no specific method of treatment.

10. *Anhedonia.* This "lack of feeling" is sometimes complained of by neurotic patients on medication and also is not infrequently noted after the psychotic patient is completely relieved of his abnormal mental content. The analeptics are moderately but not markedly successful in helping correct this side effect and one of us (E.G.) has had a few patients who seem to respond to sub-coma insulin in doses of 10 to 15 mg. daily. After the reserpine has been withdrawn for a period of several weeks, we have had some patients who were still suffering from anhedonia and who then responded well to two or three electric convulsive treatments.

11. *Depression.* There is little question that depression is seen more frequently in patients on reserpine than is prevalent in an equivalent population not on the drug. It is an open question as to whether the drug directly produces this effect or merely unveils an underlying condition which was already present. We are currently evaluating some other alkaloids of Rauwolfia-serpentina to discover whether they have this same undesirable side effect. Here again the analeptics are only moderately successful and the sub-coma insulin found useful for anhedonia is not of much help. The best treatment would appear to be electric shock therapy given PRN. Often a single treatment is sufficient to relieve the depression. As described previously, it may be that reserpine treated patients are more susceptible to developing apnea with ECT so that they must either be watched more closely or some time allowed to elapse between the giving of the reserpine and the electric shock.

12. *Suicides.* Despite the increased incidence of depression we do not believe that the rate of suicide is increased above that found in an equivalent population not on reserpine. Among the few suicides that have occurred there has been no prior evidence of depression either before or after treatment. In our own series the incidence of suicide among reserpine or chlorpromazine treated patients is no greater than among those patients not treated with either of these medications.

Psychotherapy, in the broadest sense, is indicated throughout treatment. In the early stages, and particularly if the patient goes into a turbulent phase, reassurance is some-

times of great solace. In the integrative phase, group therapy, individual psychotherapy, occupational therapy, physical and reactional therapy as well as total push should be utilized as the facilities of the institution or the patient's environment permit. A major effect of the treatment is that many patients are made amenable to therapy who previously could not be so treated.

### *Side Effects of Combined Reserpine—Chlorpromazine Therapy*

Since chlorpromazine in the doses in which we have used it[4] acts primarily to potentiate reserpine the great majority of side effects are identical to reserpine alone and most of them also occur with chlorpromazine alone. In a number of respects, however, there are differences of degree and a few differences of kind. The following side effects we find to be less marked when combined treatment is used than when reserpine alone is used:

a) generalized tremulousness
b) gastro-intestinal symptoms
c) muscular cramps, twitchings and paresthesias
d) convulsions

In the area of psychological side effects the following side effects are also reduced in degree of incidence or severity:

a) restlessness
b) turbulent phase
c) insomnia
d) anhedonic state

The side effects of large doses of chlorpromazine are also reduced with the combination and there do occur side effects associated with chlorpromazine which do not occur with reserpine alone. It is our impression that the size of the dose is irrelevant, so that even though as low as 50 mg. a day is all the chlorpromazine needed the incidence and nature of side effects (except for parkinsonism) are not different than at 1000 to 1500 or more mg.

1. *Jaundice.* It is our impression that the appearance of clinical jaundice occurs almost as soon as it can be detected by liver function tests. The custom has been to regard a rising alkaline phosphatase as indicating a potential case of jaundice when the level reaches between 25 and 30. At this point some authorities recommend that medication be discontinued. We have not experimented in sufficient detail to determine whether the patients who are not discontinued invariably go on to develop jaundice. Once jaundice has occurred it has been customary to discontinue treatment and then later restart with a good chance that the jaundice will not occur a second time. In one or two cases we have carried treatment on despite mild jaundice and the side effect has eventually disappeared without necessitating withdrawal of the drug. That this could be done with more severe cases is very possibly the case but we have not yet had the courage to try it.

2. *Skin Reactions.* These are best handled by preventing exposure to sunlight and treating topically when they are due to photosensitivity. The allergic type is treated topically without interruption of the drug therapy.

3. *Agranulocytosis*—(two cases of which we have described in detail elsewhere).[5] We believe this to be an absolute contraindication for continuing chlorpromazine. In occasional cases we have had a marked leucocytosis which dropped off when the drug was discontinued.

### *Summary of Side Effects and Their Management in Reserpine Treatment*

Twenty-five organic and a dozen psychological side effects of reserpine treatment are described along with the treatment which has been found most effective. Modifications and additions when combined reserpine-chlorpromazine treatment is used are detailed.

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# Relationship of Ability to Verbalize to Toleration of Long Term Hospitalization

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*I. Introduction:*

The high rate of irregular discharge among tuberculosis patients creates a serious public health problem which has attracted the attention of many investigators. Recently Newman and Sparer[3] on the basis of their studies reached the conclusion that the patient who leaves the hospital against advice is among other things "inadequate in language skills and lacks the verbal ability for expressing his psychological reactions." If this were established it would have important practical implications for hospital management and control. Certainly it is theoretically plausible, fitting in with current psychodynamic formulations. In view of the fact that Newman and Sparer present no substantiating data, however, it has seemed desirable to seek empirical confirmation of their position.

*II. Hypotheses:*

Newman and Sparer state that "inadequate language ability in the patients would interfere with performance on the sentence completion test" which was used by them, together with the Rorschach "to measure ability for verbalizing personal reactions." Since sentence completion data was also available in our files, it was used to check the conclusions of Newman and Sparer. These conclusions translated themselves into the following specific hypotheses with regard to test behavior:

I. that the irregular discharge group
   (a) will complete fewer sentences
   (b) will give briefer answers to those they do complete

II. that the more an incomplete sentence tends to evoke responses which express feelings and emotions related to the experience of hospitalization for tuberculosis

   (a) the fewer and briefer the responses it will elicit from the irregular discharge group

   (b) the greater will be the difference in response of the two groups.

*III. Method:*

Since all patients at the Baltimore Veterans Administration Hospital (tuberculosis) receive, two months after admission, the Madison Sentence Completion Test[1] devised by Calden for use with tuberculosis patients, a great deal of data was available which lent itself admirably to analysis of the relative verbal ability of patients completing hospital treatment as opposed to those leaving against advice.

To secure a random sample, Madison Sentence Completion Test records of all the patients from A to M who had left irregularly, were drawn from the files—a total of 38. The same number of cases was drawn from among those who had left the hospital with maximum hospital benefit. Since there were more who left "MHB," every third case was selected, covering the same range of the alphabet. The number of words used to reply to every sentence was counted and recorded for each patient. This provided the raw data to be analyzed.

The incomplete sentences were then ranked according to the degree to which they tended to evoke expressions of feelings or emotions relating to problems of hospitalization for tuberculosis. To achieve this, four psychologists and two social workers, all experienced in working with tuberculosis patients, were asked to rate each sentence on a four step rating scale having the following extreme categories:

Category No. 1. This sentence prompts expression in words, of feelings or thoughts pertinent to a problem area. Expression of such thoughts and feelings is the only really appropriate way to complete this sentence.

Category No. 4. This sentence does not prompt expression of feelings or thoughts pertinent to problem areas. Such expression would, in general, be an inappropriate way to complete it.

On the basis of the ratings obtained, forty-two of the eighty sentences on the MSCT were eliminated because there was some disagreement among the raters as to which side of the neutral point (between the emotionally evocative and the non-emotionally evocative side) they fall. There was unanimous agreement among the raters on the placement of the remaining thirty-eight sentences, relative to this neutral point.

An analysis of variance, based on the number of words used by each patient to each sentence, was the most appropriate way to test the hypotheses because it was necessary to take into account simultaneously the influence of two variables (type of discharge and degree of emotional evocativeness of each incomplete sentence). Since the variances of the sentences differed widely, Bartlett's test for homogeneity of variance, as described in Edwards,[2] was applied. The variances proved significantly different. Attempting to rectify this by transformation of the scores, which would have involved approximately 3,000 transformations, was rejected as impractical. Instead a group of sentences was selected, without reference to their content or rated tendency to evoke emotional expressions, whose variances, by inspection, appeared to fall within the same range. Barlett's test was then applied to these selected sentences with a resulting chi-square of 14.02 ($P>.95$ for 25 degrees of freedom) indicating that the variances could be considered homogeneous. Fortunately, the stimulus value of these sentences adequately covered the entire range of emotional evocativeness. A list of the incomplete sentences chosen with the average of their ratings is given in Table 1. The averaged ratings were utilized only to assign ranks on the variable under consideration.

TABLE 1

*List of Incomplete Sentences*

| Mean of Ratings* | Incomplete Sentence |
|---|---|
| 1.0 | My biggest fear |
| 1.2 | I feel like going home when |
| 1.3 | When the odds are against you |
| 1.3 | I will leave the hospital if |
| 1.5 | Having TB wouldn't be so bad if |
| 1.7 | When a patient is told he needs surgery |
| 1.7 | When someone enters my room wearing a mask |
| 3.5 | A good roommate is one who |
| 3.5 | I like a nurse who |
| 3.7 | The O. T. worker |
| 3.7 | What really helps my cure the most is |
| 3.8 | When I listen to the radio |
| 4.0 | What I like best here is |

*Mean of six ratings for each sentence based on the four point rating scale described in text.

An analysis of variance based on response to these sentences was then carried out. The data were classified according to the discharge category of the subjects (38 in each group) and by rank of the 13 sentences. The design is that suggested by Edwards[2] for repeated measurements on the same subjects.

*IV. Results and Discussion:*

The results of the analysis of variance are given in Table 2. Because the scores asso-

TABLE 2

*Summary of Analysis of Variance of the Verbal Responsiveness of the MHB and Irregular Discharge Groups to Incomplete Sentences of Varying Emotional Value*

| Source of Variation | Sum of Squares | Degrees of Freedom | Mean Squares | F |
|---|---|---|---|---|
| Between MHB and Irregular Groups | 146.92 | 1 | 146.92 | 2.45 |
| Between Subjects in the Same Group | 4437.50 | 74 | 59.97 | |
| | 4584.42 | 75 | | |
| Between Sentences | 270.87 | 12 | 22.57 | 3.45* |
| Interaction: Sentences by Discharge Groups | 90.37 | 12 | 7.53 | 1.15 |
| Interaction: Pooled Subjects by Sentences | 5814.45 | 888 | 6.55 | |
| | 6175.69 | 912 | | |
| TOTAL | 10760.11 | 987 | | |

*Significant at the .01 level of confidence. The other F values are not significant.

ciated with the different sentences and the interaction between the sentences and the discharge category were based on repeated measurements of the same subjects, the pooled subject by sentence interaction mean square was used as the error term in F ratios involving these sources of variance.

From Table 2 we can draw the following conclusions:

1. There is no significant overall difference between the patients receiving regular, MHB discharges and those leaving irregularly in ability to verbalize reactions to their hospital experiences, as measured by their response to selected items from the Madison Sentence Completion Test. Hypotheses Ia and Ib are therefore not supported by this data.
2. As might be expected, the analysis reveals that the patients in both groups respond differently to the various selected sentences. This means only that there are statistically significant individual differences in the power of the sentences to evoke responses, which hold over both groups. Hypothesis IIa implies that these differences are related to the degree of emotional stimulus value of the incomplete sentence. Whether this suggested relationship holds, can be seen from the rank order correlation coefficients between rank in emotional stimulus value and rank in mean number of words responded. For both groups combined, rho=—.39, for the MHB group rho=—.34, for the irregular group rho=—.35. The negative sign on the coefficients indicates that the direction of the relationship is contrary to that predicted by Hypothesis IIa. In short, the more an incomplete sentence tends to elicit expression of emotional reactions to hospitalization the greater the verbal response in both groups, with negligible difference between them. Therefore Hypothesis IIa is not supported.

   Further evidence against Hypothesis IIa is found in the rank correlation of the difference between the groups in number of patients responding to each sentence and the tendency of that sentence to evoke emotional expression. Rho=.10 which is quite negligible. This carries more weight in view of the fact that there is no particular relationship between the number of patients in each group who responded to each sentence, rho=.13.
3. There is no significant interaction between the tendency of the sentence to evoke expression of emotion and the discharge category. This means that as the incomplete sentences increase in their ability to evoke expressions of emotion, there is no corresponding significant increase or decrease in the difference in the verbal responsiveness of the two groups. Hypothesis IIb is, therefore, not supported.

Certain other evidence on the points at issue may also be marshalled. The Mann-Whitney test, applied to rankings of the patients according to the total number of responses each gave over the entire 80 sentences, disregarding their stimulus value, indicates that the distributions do differ significantly with the irregular discharge group tending to complete fewer sentences ($P < .036$ for a one-tailed test of the hypothesis). While this supports Hypothesis Ia, consideration of other evidence leads to an interpretation essentially different from that of Newman and Sparer. First, the analysis of variance provided contrary evidence. Second, the number of words used overall by each group, when allowance is made for the number of sentences not completed at all, do not differ significantly. Third, the frequency distributions of the total number of sentences responded to show the familiar J curves of conformity. These curves, which are typical in data describing conformity of groups to various social institutions, also describe the response of the patients to the Madison Sentence Completion Test, with the frequency curve for the irregular group showing less conformity. This suggests that the significant difference in distributions reflects, in the irregular discharge group, a tendency to non-conformity, rather than to inadequacy in language skills or to inability to express emotional reactions to hospitalization. That is, the patients who leave against advice conform less well to the request that they fill out the sentence completion form just as they conform less well to the expectation of society that they remain in the hospital until fully treated.

In order to test this interpretation of the patients' behavior in terms of conformity or cooperativeness, it was decided to investigate the relationship between complete refusal to cooperate in taking the Madison Sentence Completion Test and irregular discharge. To do this all patients who had been given an opportunity to take the test were classified as to whether they had made some effort to fill out the form or whether they had rejected it completely. Excluded from this latter group were those who had legitimate physical reasons for not taking the test (too ill, blind, paralyzed, etc.). Also excluded from the comparison were patients whose discharge from the hospital did not

fall into either the MHB or irregular discharge category (for example, patients who died in the hospital or who were transferred to other institutions). The classification of the remaining 570 patients according both to type of discharge and acceptance or refusal of the test is given in Table 3. (The N for those accepting the test differs from that implied in the samples drawn for the analysis of variance because this data was taken from the files nine months after the samples were drawn.)

TABLE 3

*Classification of Patients According to Discharge Category and Refusal or Acceptance of the Madison Sentence Completion Test.*

| *Test Behavior* | *Type of Discharge* | | |
|---|---|---|---|
| | MHB | Irregular | Total |
| Accepted | 375 | 101 | 476 |
| Refused | 57 | 37 | 94 |
| TOTAL | 432 | 138 | 570 |

The chi-square for the relationship of cooperativeness in taking the test to type of discharge received is 14.08 which is significant at beyond the .01 level of confidence for one degree of freedom. From this we may conclude that there is indeed a reliable relationship between these variables. The degree of this relationship is reflected in the fact that among those patients who take the test the irregular discharge rate is 21%, whereas among those patients who refuse the test the irregular discharge rate is 39%.

This result clearly supports the hypothesis derived from the previous analysis, *viz.* that non-conformity is an important factor underlying irregular discharge. Further investigation is, of course, necessary, utilizing various measures of "non-conformity" to clarify more fully its meaning in this hospital situation, and to apply more refined measures to the prediction not only of type of discharge, but to the length of hospitalization likely to be tolerated before the patient leaves against advice.

*V. Summary and Conclusions:*

An analysis of variance of the number of words used to complete selected sentences having different degrees of emotional evocativeness was done, using data from the Madison Sentence Completion Test for tuberculosis patients, for a group of patients (N=38) who left the hospital irregularly, and a group of patients (N=38) who completed hospital treatment to the satisfaction of the medical staff.

The results offer no support for the conclusion of Newman and Sparer that the patient who leaves against advice is "inadequate in language skills and lacks the ability for expressing his psychological reactions." On the other hand there is some evidence that a general tendency not to conform may be operative among those who leave irregularly. More intensive investigation of this finding is, of course, in order.

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# Ritalin and Serpasil in the Treatment of Parkinson's Disease

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The treatment of parkinsonism is at times extremely difficult because of the effects of the disease, both directly and indirectly, on the emotions and mood. The drugs used in this condition are often pushed to tolerance in order to get full therapeutic effect, creating clouding of consciousness and reduction in the efficiency of the perceptive and aperceptive faculties. This results in an increase in anxiety and perplexity with a reactive depression being a not uncommon concomitant. The psychic disorder often produced by the disease and its treatment invariably aggravates the organic symptoms which in turn aggravate the psyche initiating a vicious circular reaction which is often the main deteriorative force in this disease.

In the past, the use of barbiturates, bromides, chloral, and other sedatives has been of little use as adjuvant therapy in regard to the problem stated. With the advent of Thorazine, Serpasil, and the newer tranquilizers, a definite step has been taken in this direction. However, their use has been disappointing in several respects. Thorazine, although useful in ameliorating the insomnia found in Parkinsonism, is of little improvement over the usual sedatives in combating the psychic symptoms found as aggravating influences. From my own experience, it often causes an intensification of the most annoying symptoms of clouding of mentation and visual disturbances found as side effects with the anticholinergic drugs used in treatment. Serpasil has proven to be of value as a tranquilizer in this condition, but it often has depressive side effects. The use of an adjunct to combat the depressive tendency found in using Serpasil would appear to be helpful in creating a tranquilizing combination. This paper concerns itself with the use of such a preparation. The drug used with Serpasil is Ritalin. Ritalin has properties which make it of value, not only in combating the possible depressive effects of Serpasil, but also has independent adjunctive therapeutic advantages in that it potentiates the parasympathicolytic action of other drugs and may have an independent action in ameliorating tremor. A summary of its most outstanding pharmacological properties is presented below.

Ritalin is chemically methyl alpha phenyl-2-piperidineacetate hydrochloride. It is water soluble and forms two racemates differing greatly in intensity of effect. The active racemate is the meso form which is known in the pure state as Ritalin. The substance has a psychomotor stimulating effect proven in animals by general restlessness, a coordinated increase in mobility, and an urge to eat or gnaw without becoming snappish or aggressive. The reaction noted depends on the dose, the individual animal, the species, and subsides leaving only fatigue. If given increased doses, an ataxic gait and tonic clonic convulsions can be produced. The increase in mobility observed can be increased by atropine and decreased by Regitine. Phenobarbital blocks the analeptic action of Ritalin, but under certain conditions may stimulate the increased motoricity. Pentothal has little influence on this analeptic action with choral, urethane, and other barbiturates intermediate as regards this effect. Serpasil has a definite blocking effect on the increased motor function induced by Ritalin.

Ritalin stimulates respiration and also causes a prolonged moderate rise in blood pressure with little or no effect on peripheral circulation. There is a tachyphylactic reaction in this regard. Its peripheral sympathicomimetic reaction is not marked. Ritalin has little or no effect on appetite, speeds up the normal EEG rhythms, and reduces the Parkinson-like tremor produced in monkeys by electrical coagulation of certain areas of the brain stem. As regards its effect, the drug may be classified somewhere between amphetamine and caffeine.

As can be seen, Ritalin is directly antagonistic to the psychomotor depression often

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caused by Serpasil; it potentiates the action of the parasympathicolytic drugs used in Parkinsonism, and apparently can directly ameliorate tremor. Thus, when used with Serpasil as a tranquilizer, the combination should be of definite use as an adjunct in the treatment of this condition.

Using 0.25 milligrams of Serpasil and 5 milligrams of Ritalin t.i.d. to q.i.d., in conjunction with the basic drugs used in treatment, a definite improvement in the clinical condition of 90% of 18 institutionalized and private cases suffering from post-encephalitic parkinsonism was noted. All the patients had been on therapy prior to its use, and had reached a plateau as regards stabilization of clinical symptoms. With the adjuvant treatment, an improvement in mood, psychomotor drive, and mental functioning was noted along with reduction in rigidity, tremor, occurrence of oculogyric crises and salivation. Patients who had presented acute problems in management due to anxiety and agitation and its indirect influence on the clinical effects of the disease were improved to the point of patient becoming cooperative and happy with the resultant amelioration of subjective and objective symptoms. Several patients complained of severe nausea and severe nasal stuffiness and were taken off the drug. A week to ten days' administration was often necessary before seeing any improvement and its effects lasted for approximately this time when the patients were taken off the drugs. Placebo medication did not reproduce the improvement. Raising or lowering either the Serpasil or Ritalin dose of the combination may accentuate one or the other drug effect.

In summary, I wish to state that this preliminary study would suggest that this combination, i.e., Ritalin and Serpasil, has a place as an adjunct in the treatment of parkinsonism and should be given a trial in those cases where psychic effects, such as anxiety, agitation, depression, and decreased psychomotor activity are causing an aggravation of the organic clinical symptoms with the pernicious circular effect this aggravating influence is capable of setting in motion.

*Author's Note:* Ritalin and Serpasil used in this study are manufactured by Ciba Pharmaceutical Products, Inc., Summit, N. J.

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# Eastern Psychiatric Research Association Proceedings

DR. CHARLES BUCKMAN, *Chairman*

## Management of Children and Adolescent Girls

DR. OLGA VON TAUBER

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Child psychiatrists, particularly those in private practice often encounter emotionally disturbed and neurotic children. Existence of true psychosis in children is denied by many experts, but authorities such as Doctors Bender and Kanner have described the syndrome of childhood schizophrenia and early autism. Psychotic episodes incident to organic brain disease are known to occur. Psychosis in childhood, however, is comparatively rare but contributes to the number of children admitted to institutions.

Read at the Meeting of Eastern Research Assn., held at Kings Park State Hospital, Apr. 5, 1956.

At Kings Park, we receive children representing all categories of mental disorders occurring in childhood. For the past two years the majority of girls admitted were children with organic brain damage.

The life histories of our children regardless of diagnosis, show the characteristic combination of factors which are considered important genetic and dynamic determinants for their behavior deviations. Deprivation of love, parental neglect, cruelty, inconsistency are a frequent group of factors. Overprotection, parental ambition beyond the child's capacities, sibling rivalry form another group of factors detrimental to healthy development of the personality of the child. Children exposed to such traumatic experiences have never felt secure and therefore they have become distrustful. When with other children and adults they sense the difference and feel even more rejected and lonely. Aggression and resentment become a means of compensation. In

their vain struggle for recognition and self-assertion constant failure perpetuates itself. One wrong step leads to a greater mistake and in some cases, to serious delinquent acts. These children in whom environmental situations can be made largely responsible for the behavior difficulties are the so-called primary behavior disorders. If not helped in time, they may grow up to full-blown psychopaths.

Infectious illnesses, head trauma, are the factors most frequently responsible for organic brain damage in children. The behavior difficulties in these children are easier to understand and sympathy is more readily extended to them than to the so-called primary behavior disorders, where the cause is not so obvious. Mental retardation, convulsions, frequently associated, are further handicaps for the brain damaged child. Frustration, rejection and isolation at home, in school and in the community lead to similar conduct disorders as observed in environmental behavior problems. In contrast, the schizophrenic child tries to solve his conflicts by withdrawal in a world of phantasy. This child may range from a state of complete indifference and passivity to sudden explosive, impulsive behavior. The autistic child as described by Dr. Kanner, is considered only a sub-group of schizophrenia. Finally, we observe children who show the syndrome of childhood schizophrenia, as Dr. Bender sees it, with hyperkinesis, the characteristic play-pattern and certain vaso-vegetative disturbances. Quoting Dr. Bender, "Disturbances in identification processes, that is, the difficulty of identifying oneself and thereby relating to the rest of the world, is the essential psychological problem. The resulting anxiety is a reactive problem and the symptom formation is related to both."

We are frequently asked if the emotionally disturbed child with behavior difficulties should be considered mentally ill. I do believe that such children are mentally ill. Probably when functioning is interfered with to such a degree that adjustment at home, in school and in the community is no longer possible, the more accurate term would be psychotic. In a child, even more than in the adult, the distinction between emotional difficulties, neurotic behavior and psychotic manifestations is one of degree rather than of kind and essence. Another question frequently asked whether these children belong in state hospitals. This is not the proper type of institution, but there are few other facilities in the communities for them.

Kings Park was not originally equipped to deal with children and we still meet with physical and other limitations in our set-up. To organize a therapeutic and educational program designed to reach all these children is extremely difficult. We have tried to create in the girls' unit what I would like to call a therapeutic atmosphere. We have attempted to be permissive and directive at the same time. Our employees have been selected as carefully as possible choosing people who are warm and sympathetic and have understanding of the problems. To give the child affection, security and a feeling of belonging, which in many cases he has never experienced, was an important goal. Our program was planned to stimulate the child by achievement and acceptance in the group. It includes school which alternates with arts and crafts classes and there is a variety of recreational activities. We have started a Girl Scout program and several volunteer groups from nearby communities assist us.

The work of these volunteer groups could be considered a social relationship group therapy. By giving gifts, treating the children, by group playing, it has been possible to include at times even the more disturbed and aggressive children who could not be reached otherwise.

Group therapy was done with the adolescent girls with true behavior disorders. The focus of the group was rebuilding the reaction patterns in the direction of more acceptable behavior. The leaders of the groups were chosen by the girls, the writer limiting herself mainly to giving interpretation. I believe that the group presented to these girls, a miniature of a non-threatening society. There was no need for aggression which was the usual defense mechanism for these girls. Play therapy on a limited scale was used for the small children. Electroshock therapy was rarely applied and a few selective cases of adolescent girls were referred to the insulin service. More recently, we gave Thorazine and Serpasil to the hyperactive, organic and primary behavior disorders including a few epileptics who showed marked emotional overlay. Our results with Thorazine were better than with Serpasil.

Here are figures to illustrate the relationship of returns to the community and of admissions, etc.:

| Annual Period (Beg. March) | 1952 | 1953 | 1954 | 1955 |
|---|---|---|---|---|
| Number of children admitted (under 16 years of age) | 42 | 20 | 9 | 12 |
| Number of children placed on convalescent care | 73 | 32 | 21 | 15 |
| Number of elopements | 9 | 0 | 0 | 0 |
| Number of returns from convalescent care | 12 | 3 | 6 | 5 |
| Number discharged outright | — | 1 | 1 | — |
| Discharged and certified to Willowbrook State School | — | — | 4 | 3 |

The mental-defective child, once the psychotic episode has cleared can be transferred to a state school.

I believe that the comparatively high number of returns to the community is due to the fact that these children for the first time in their lives, found acceptance, companionship and had the opportunity to form more dependable adult relationships. The program they participated in was geared as much as possible to their individual needs, abilities and special interests.

Because of lack of time, the children did not have sufficient intensive psychotherapy, did not in fact, receive too much individual attention. Perhaps it would be correct to assume that individual needs were compensated by a feeling of belonging and the satisfaction which is derived from the capacity to adapt and conform.

# BOOK REVIEWS

**DOCTOR AND PATIENT AND THE LAW: By Louis J. Regan, M.D., LL.B., Member State Bar of California, Professor of Legal Medicine, College of Medical Evangelists; Clinical Professor of Forensic Medicine, School of Medicine, University of Southern California. Cloth. Pp. 716. $12.50. Third Edition. The C. V. Mosby Company, St. Louis, Mo. 1956.**

In the conduct of his profession the physician's obligations are bound up with many and involved aspects of legal responsibilities both in respect of his patient and of the locality wherein he engages in his professional practice. To avoid the risk of involvement and to keep clear of legal breaches, it is prudent for the physician to consider these contingencies by acquiring a general understanding of the principles of medicine and their interrelation with the many-sided principles of legal precepts.

In furtherance of such guidance, the author has outlined the medicolegal principles and has methodically illustrated by the use of legal citations and examples for clarification, the distinguishing propositions of law and their application to principles governing conduct, action and procedures where medical comportment calls for conformity with the law.

Legal medicine can contribute much toward the administration of justice and by virtue of its nature, involves the physician's scientific contributions to the studies of forensic pathology and psychiatry. The physician's interests equally involve a consideration of the problems flowing from his peculiar duties, privileges and responsibilities which legally devolve upon him and his relationship to his patient.

The book deals with the latter phases and provides in plainly couched language an integrated and knowledgeable understanding of the intricacies of the statutes and related court decisions in both State and Federal jurisdistions. Herein the author has included the role which falls to the physician in situations involving adoption, abortion, commitment, artificial insemination, workmen's compensation, narcotics, the reporting of vital statistics, communicable diseases, etc. The book allots considerable space to the intriguing topic of malpractice and the author ingeniously outlines the patient's interest therein, and the likelihood of the physician's involvement in the occupational hazard of an unjustifiable suit for damages. The author concludes with a malpractice program which offers practical guidance to assist the physician toward a recognition of his duties, limitations and what may be expected of him, and toward an effective understanding of how to govern and protect himself under the law.

*Lewis J. Siegal, M.D., LL.B.*

**BING'S LOCAL DIAGNOSIS IN NEUROLOGICAL DISEASES: By Webb Haymaker, M.D., Professor of Neurology, Georgetown University School of Medicine, Washington, D.C. Cloth. Pp. 478 with 28 illustrations. The C. V. Mosby Company, St. Louis, Mo. 1956.**

In its present fourteenth revision, this edition is a markedly amplified presentation of the original treatise of Bing's compendium of clinical neurology. Although it continues to retain its didactic format it remains weighted with unspeculative matter.

The book combines the fundamentals of neuroanatomic principles wih those of clinical case material, and in a direct manner appraises through the clinicopathologic approach, the localization of brain and spinal cord lesions.

The illustrations, some of which have been drawn in color, are in excess of 200, are replete with photographs of clinical cases as well as of pathological case material. The volume concludes with two chapters, one of which is on x-ray localization of intracranial lesions through plain film as well as pneumoencephalographic and angiographic media; and the reproductions of a variety of neoplastic conditions merit special mention because of their clarity and vivid photographic portrayal. The closing chapter discusses the present concept of the influence of cerebral tissue activity upon electroencephalographic tracings and evaluates their correlative approach to clinical manifestations, in the light of their use in cerebral lesion localization.

The bibliography, which precedes a well systematized index, is sufficiently lavish to serve the most intent investigator of the subject. The book well merits the perusal of the specialist as well as of the general physician concerned within the domain of neurodiagnosis.

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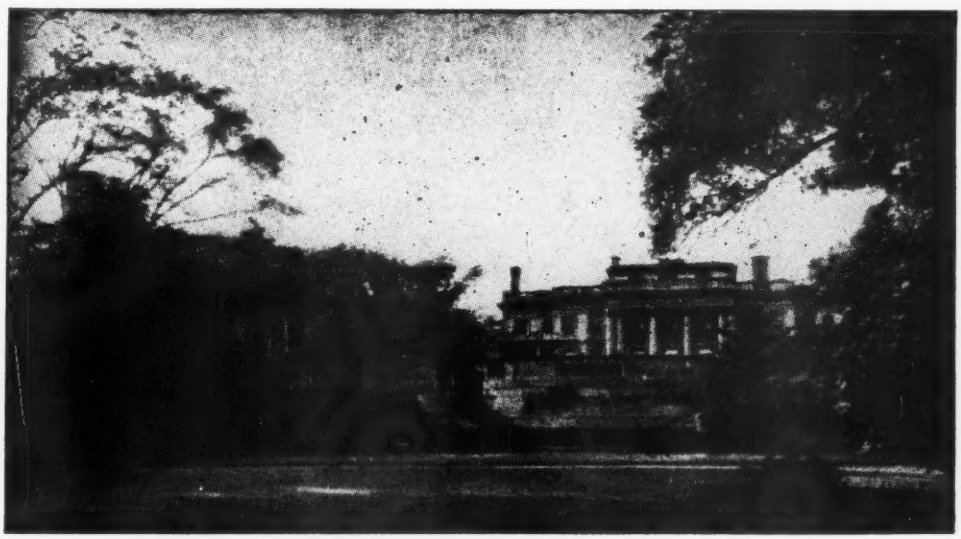

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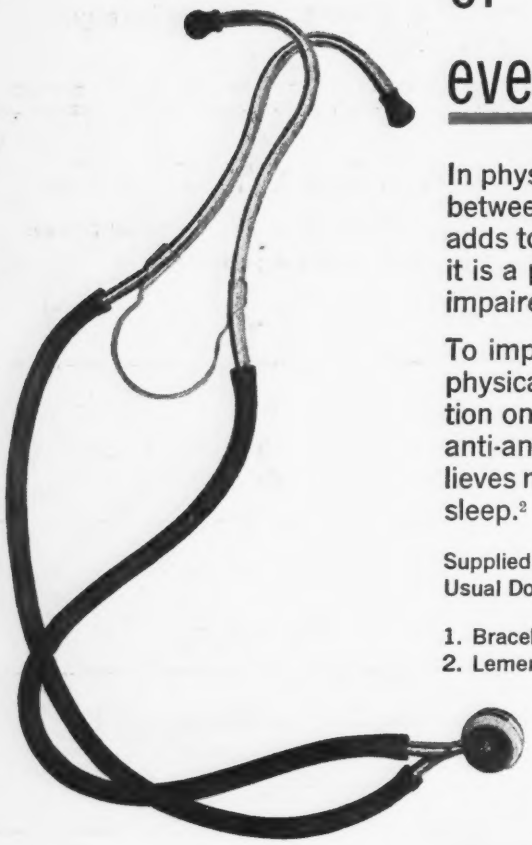

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